Significant Left Main Stenosis following Asymptomatic Dissection during Coronary Arteriography

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The natural history of minimally symptomatic or asymptomatic iatrogenic coronary dissection is not well-defined. While generally considered a serious and life-threatening complication of coronary arteriography, there have been some reported cases of spontaneous resolution without residuals. We describe a case demonstrating that coronary artery dissection, even when initially asymptomatic, may later progress and result in significant or complete coronary occlusion. The implications of this are discussed.

Coronary artery dissection is a well-recognized but fortunately infrequent complication of left heart catheterization and coronary arteriography. The clinical consequences of coronary dissection most often become apparent immediately or shortly following the procedure and vary widely from no symptoms to transient angina to serious dysrhythmia, myocardial infarction, cardiogenic shock, and death.14 Even when the patient is asymptomatic at the time of dissection, the subsequent course is fraught with danger. We describe a patient who, although asymptomatic following left main coronary dissection, subsequently had a marked exacerbation of chronic exertional angina. Repeated coronary arteriography demonstrated significant narrowing of the residual left main coronary artery by an enlarged false lumen originating at the site of previous dissection, which appeared to be responsible for the clinical deterioration.

CASE REPORT

A 64-year-old man with severe peripheral vascular disease underwent elective cardiac catheterization for angina. Coronary arteriography by the brachial artery approach using a Sones catheter revealed total occlusion of the right coronary artery and a 60 percent stenosis of a left circumflex marginal. No other significant lesions were seen, and the left main coronary artery appeared normal. During the course of the procedure, a small intimal flap was raised, and a highly localized subintimal contrast stain appeared in the left main trunk at the site of intimal penetration (Fig 1A). However, the patient remained hemodynamically stable and clinically asymptomatic, without angina or ischemic ECG changes. Following hospital discharge, and over the ensuing weeks, the patient had worsening of exertional angina despite increased use of medications with nitrates and β-blockers. Because of progression and severity of angina, causing functional class 4 limitation in physical activity, the patient was readmitted for a repeat catheterization. Brachial artery approach and a No. 6F Amplatz catheter were utilized during the second procedure. Before entering the left orifice, injection of contrast into the left sinus of Valsalva indicated a significant abnormality of the left main trunk. After entering the left main orifice, injection of contrast first visualized a false lumen 0.8 to 1.0 cm long and as wide as the untapered diameter of the No. 8F Amplatz catheter (2.67 mm; Fig 1B). As dye refluxed out of the false channel into the aortic sinus, the residual lumen was seen and the dissected intima clearly identified (Fig 1C). No attempt was made to selectively cannulate the residual true lumen, and the procedure was terminated without complication. In view of the patient's severe angina and the left main disease demonstrated, coronary bypass surgery was undertaken, from which the patient recovered with marked functional improvement.

DISCUSSION

Catheter-induced coronary dissection is an infrequent

FIGURE 1A. Cineangiographic frame showing RAO left coronary angiogram. Tip of Sones catheter (arrow) in opposition to small intimal flap in left main coronary artery. B. Shallow LAO view of catheter tip (arrow) within false lumen which fills with angiographic contrast at very onset of coronary injection. C. Later phase of same injection as 1B, showing reflux of angiographic contrast from false lumen into left coronary sinus; subsequent faint opacification of coronary system as contrast enters residual true lumen. Dissected intima clearly delineated (arrow) and residual lumen appears significantly narrowed.
complication of coronary arteriography, with a variable incidence in large series of 0.013 to 0.35 percent. 4,5 The extent of dissection may remain highly localized to the site of intimal penetration, extend along the length of the coronary artery, or even progress retrograde into the sinus of Valsalva and aortic root. 6,7 Coronary dissection may limit blood flow to a variable extent as a result of the degree to which subintimal hematomas or the intimal flap obstructs the true coronary lumen. Episodes of readily reversible ST segment elevation following dissection suggest that superimposed coronary spasm may also occur in some instances. 4

The immediate consequences of dissection are well-recognized and range from no symptoms or transient angina to unstable angina, serious ventricular dysrhythmia, myocardial infarction, cardiogenic shock, and death. While symptoms frequently develop in association with dissection, a surprising proportion of patients may be asymptomatic. Weiner et al 8 noted no symptoms associated with dissection in five of 12 cases, representing experience with the largest group of iatrogenic coronary dissections reported to date. Regardless of their symptomatic status at the time of dissection, the follow-up course of these patients is unpredictable.

Although cases have been reported that appeared to follow a benign course ending in spontaneous resolution, 4,9 there have also been documented instances of delayed acute progression resulting in complete coronary occlusion. 7

The present case represents further demonstration that asymptomatic coronary dissection may follow a malignant course. In our case, a persistent false lumen appeared to enlarge and create significant left main stenosis, exacerbating chronic angina. In addition, the persistent false lumen with an intimal flap exposed to antegrade blood flow posed the threat of sudden acute progression, resulting in infarction or sudden death. Because of unpredictable course and potential for sudden catastrophic coronary occlusion, we suggest that any dissection of the left main coronary artery should best be handled as a surgical emergency and the patient undergo urgent coronary bypass operation.

REFERENCES
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Successful Repair of Criss-Cross Heart Using Modified Fontan Operation*

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A five-year-old girl with concordant crossing of artioventricular connection, hypoplastic tricuspid valve, straddling mitral valve, 1-transposition of the great arteries, and other anomalies was operated upon. Modified Fontan operation was beneficial for such a complex cardiac anomaly.

The criss-cross heart is a rare cardiac anomaly characterized by apparent crossing of the artioventricular blood streams in the frontal view. 10 This paper describes our experience using modified Fontan operation in a patient with concordant criss-cross artioventricular connection.

CASE REPORT

A girl, five years and four months of age, was admitted to Chiba Cardiopulmonary Center with cyanosis and clubbing. A grade 4/6 ejection murmur was heard at the left sternal border with thrill. The liver was palpable 3 cm below the right costal margin at the middiavascular line. The ECG showed a regular sinus rhythm with a rate of 120 beats per minute, a QRS axis of +118°, and marked clockwise rotation. The chest x-ray film revealed a cardiothoracic ratio of 49 percent and a straight left upper cardiac border. At cardiac catheterization, the catheter was inserted into the left-anterior ventricle, which had the morphology of a right ventricle, through the right atrium and tricuspid valve (Fig 1, a and b). The sinus portion of the right ventricle was right and small, the infundibular portion was left and large (D-loop). The catheter was also inserted into the right-posterior ventricle, which had the morphology of a left ventricle, via an atrial septal defect, left atrium and mitral valve (Fig 1, c and d).

Therefore, artioventricular connection was criss-cross in anteroposterior view. The mitral valve was considered to straddle the interventricular septum through the large ventricular septal defect. The pressure of each ventricle was 85 mm Hg in systole. The great arteries were transposed (Fig 1). The diagnosis of the catheterization was situs solitus, concordant crossing of artioventricular connection, ventricular septal defect, 1-transposition of the great arteries, subpulmonary stenosis, and atrial septal defect. The straddling mitral valve and hypoplasia of the tricuspid orifice were not well evaluated at this time.

On Jan 20, 1981, she was operated upon through median sternotomy. The operative findings were the same as the preoperative diagnosis, including a very hypoplastic tricuspid valve with diameter of only 5 mm and straddling mitral valve. The main pulmonary artery was anastomosed to the right atrial appendage after closure of the atrial septal defect with a Dacron patch and direct suturing of the orifice of the tricuspid valve. A satisfactory hemodynamic state was obtained after the operation without catecholamines.

Postoperative catheterization was performed on Feb 12, 1981. The mean pressure of the right atrium was 16 mm Hg, and there was no pressure gradient between the right atrium and pulmonary artery. The trigram showed good pulmonary flow. She is now well one and one-half years after operation and is without cyanosis, cardiomegaly, or hepatomegaly.

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